

INTESTINE INFECTIONS:

TYPHOID FEVER:

They include - tuberculosis, typhoid, colonic disease, peptic ulcer

Typhoid - caused by *Salmonella typhi* & *paratyphi*.
- colonizes in the gall bladder

Mode of transmission - person to person contact.

Ingestion of contaminated food & water

Incubation period - 7-14 days.

PATHOGENESIS: (They can survive in gastric Acid of stomach)

Events →

During which asymptomatic period

Attach to the microvilli

penetrates the ileal mucosa.

↓
Salmonella phagocytosed by the macrophages (Peyer's patches)

multiplying within the macrophages are carried to the mesenteric lymph nodes

• During bacteraemia → are seeded in many organs.

• Bile is good culture Medium for the typhoid bacillus, multiply in the gall bladder.

→ Peyer's patches and lymphoid follicles - bacilli located in these.

↓
oral typhoid ulcers:

MORPHOLOGY: lesions
└─> intestinal
└─> extraintestinal

GROSS:

Location → terminal ileum.

App → Ulceration & necrosis of mucosa over Peyer's Patches minor produce typhoid ulcers.

Characteristics of typhoid ulcer:

Orientation: The ulcers are oval and oriented along the long axis of bowel.
Base of the ulcer → black due to elongated mucosa.
Edges → Raised.

MICROSCOPY: Oval ulcers.

Lamina propria - Macrophages, lymphocytes, plasma cells, Neutrophils

Extraintestinal lesions:

→ Typhoid nodule → formation of focal granuloma - Typhoid nodules
Aggregates of Macrophages

→ Abdominal muscles → Zenker degeneration → necrosis of skeletal muscle.

→ Gall bladder → Typhoid cholecystitis.

→ Mesenteric lymph node.

CF → bloating, nausea, vomiting, diarrhea, AP, Rose spots, spleen.

Complications → Perforation of ulcer, Hemorrhage from the ulcer, Carcinoma, etc.

LB test → Blood culture, Widal test, TLC, Urine culture.

SHIGELLOSIS - BACILLARY DYSENTERY:

Ulcerating lesions of large intestine.

Necrotizing infection of the distal small bowel colon caused by shigella.

most common - bloody diarrhea.

→ Shigella - Unencapsulated, non motile, AGBB,

Mode of infection → Human are the only natural reservoir.

MoP → Fingers.

PATHOGENESIS:

most virulent → (Resistant to Action of Acid in the stomach)

penetrate Intestinal Mucosa taken up by Macrophages
phagocytosed by Macrophages



Inflammatory Rx → loosens the intercellular bases of damaged surface epithelium leading to superficial ulcers.



Produce toxin

MORPHOLOGY:

Not prominent in the left colon mainly Rectosigmoid Area
lesions are continuous & diffuse.

→ ulcer → edema, ulceration, friable granular, hemorrhagic mucosa
↓ to the long Axis of the colon.

MICROSCOPY:

Early stage → small Aphthous ulcer,

Adv stage → Necrosis of epithelial cell covered by purulent exudate.

ulcer may extend into muscular mucosa.

CP → Bacillary dysentery is the most common cause of bloody discharge.

Complication → Reiter syndrome

Hemolytic uremic syndrome (HUS) due to toxins

ANEBIASIS:

Infections caused by the protozoan *Entamoeba histolytica*.

3 stages:

1) Trophozoite stage → unshaped ovoid organism stained by PAS.

2) Preys stage

3) 1st stage: infecting stage

Source of infection → Human as only known Reservoir for *E. histolytica*.

PATHOGENESIS: